The etiology of childhood specific phobia: A multifactorial model

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The Etiology of Childhood Specific Phobia: A Multifactorial Model

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Specific fears of the dark, animals, blood, heights and so on are common in childhood (see, e.g., King, Hamilton, & Ollendick, 1988), yet most of them are short-lived and dissipate within months (Bauer, 1976; Ferrari, 1986). However, in some children, specific fears become severe and invalidating in the sense that they interfere with normal functioning. In these cases, the diagnosis of specific phobia should be considered (see the latest edition of the *Diagnostic and statistical manual of mental disorders* [DSM-IV] published by the American Psychiatric Association in 1994).

The present chapter describes a multifactorial model of the etiology of (childhood) specific phobias. The model that is sketched draws heavily on ideas and concepts proposed by other authors (e.g., Craske, 1997; Taylor, 1998; King, Gullone, & Ollendick, 1998) and attempts to integrate the empirical material that has accumulated over the past years. It rests on the following observations:

1. The majority of children display normal developmental fears that wane with the passage of time.
2. A minority of children have a genetic vulnerability that predisposes them to develop maladaptive fears.
3. This genetic vulnerability manifests itself in certain behavioral patterns.
4. Discrete learning experiences interact with normal developmental fears and genetically linked behavioral patterns to produce extremely persistent fears that culminate in specific phobias.
5. Once a specific phobia exists, it is maintained by cognitive biases.

The sections that follow are organized around these points. The chapter concludes with a critical discussion of the model and its concurrents.

Normal and Abnormal Childhood Fears

Normal Fears

Studies on childhood fears have relied predominantly on surveys that list a broad range of potentially fear-provoking stimuli. A widely used instrument

for this purpose is the revised version of the Fear Survey Schedule for Children (FSSC-R; Ollendick, 1983). The FSSC-R asks children to indicate on three-point scales ("none", "some", "a lot") how much they fear specific stimuli or situations. FSSC-R surveys indicate that "normal" children and adolescents report a surprisingly large number of fears. For example, Ollendick, King, and Frary (1989) found an average of 14 fears reported by American and Australian youths aged 7–17 years. There are good reasons to believe that this number is quite similar across different nationalities (e.g., Ollendick, Yang, King, Dong, & Akande, 1996), that girls exhibit significantly more fears than boys (Ollendick et al., 1989), and that younger children endorse more specific fears than older children (Ollendick et al., 1989). A recent study by Gullone and King (1997) showed that, although children's level of fearfulness generally decreased over a 3-year period, initial fear scores were good predictors of follow-up fear scores—suggesting a trait component of fearfulness.

Studies employing the FSSC-R consistently indicate that prevalent fears of children are nearly always related to dangerous situations and physical harm. More specifically, FSSC-R studies revealed the following rank order for common childhood fears: (1) not being able to breathe; (2) being hit by a car or truck; (3) bombing attacks; (4) getting burned by fire; (5) falling from a high place; (6) burglar breaking into the house; (7) earthquake; (8) death; (9) illness; and (10) snakes (e.g., Ollendick et al., 1989; Ollendick, Yule, & Ollier, 1991). It is obvious that this rank order depends critically on the fear items listed by the FSSC-R (see, for a similar argument in the adult literature, Kirkpatrick, 1984). Recently, Muris and colleagues (Muris, Merckelbach, & Collaris, 1997; Muris, Merckelbach, Meesters, & Van Lier, 1997) examined the prevalence of common childhood fears by employing a different approach. In their studies, fear rank orders were obtained by asking children what they feared most without specifying items a priori. Results indicated that the fear rank order based on this "free option" method substantially deviates from that produced by the FSSC-R surveys (see Muris, Merckelbach et al., 1997). For example, whereas the free option method suggests that top intense fears pertain to animals (in particular, fear of spiders), FSSC-R studies indicate that top intense fears have to do with danger and death.

Developmental Patterns

A number of studies have investigated the developmental course of childhood fears (see, for a detailed review, Marks, 1987). In this context, one of the most cited studies is that by Bauer (1976) who asked 4- to 12-year-old children to specify what they feared most. Results showed that 74% of the 4- to 6- year-olds, 53% of the 6- to 8-year-olds, but only 5% of the 10- to 12-year-olds reported fears of ghosts and monsters. In contrast, only 11% of the 4- to 6-year- olds, but 53% of the 6- to 8-year-olds, and 55% of the 10- to 12-year-olds reported fears of bodily injury and physical danger. Similar results were obtained by Muris, Merckelbach, Gadet, and Moulaert (2000)

who investigated the content of fear among 4- to 12-year-old children. Their study also illustrates that specific fears are fairly common among 4- to 6-year old children (71.0%), peak between the ages 7 to 9 (87.0%), and then decline in 10- to 12-year-olds (67.8%). Taken together, these results imply that the content of fears changes as children grow older and that fears are most prominent in the 7–9 years age range. In addition, the developmental changes of specific fears follow a highly predictable course, a phenomenon that has been termed "the ontogenetic parade of normal fears (Marks, 1987, p. 109).

Children's cognitive capacities are an important determinant of the ontogenetic parade of fears. This is not surprising given the fact that fear originates from threat and threat has to be conceptualized. Conceptualization, in turn, depends critically on cognitive abilities (e.g., Vasey, 1993). Thus, at very young ages, specific fears are directed at immediate, concrete threats (e.g., loud noises, loss of physical support, separation of mother). As cognitive abilities reach a certain maturational stage, specific fears become more sophisticated. For example, at 9 months, children learn to differentiate between familiar and unfamiliar faces and, consequently, fear of strangers becomes manifest. Following this, fears of imaginary creatures occur and these are thought to be intimately linked to the magical thinking of toddlers (Bauer, 1976). Fear of animals also develops during this phase. These fears are believed to be functionally linked to the increasing mobility of the child. That is, fear of animals would have survival value, as it would protect the mobile child from predators (Öhman, Dimberg, & Öst, 1985). From age 7 onwards, children are increasingly able to infer physical cause–effect relationships and to anticipate potential negative outcomes. These cognitive changes probably broaden the range of fear-provoking stimuli and, thus, fear of blood and injury, natural events, and social encounters develop (e.g., Vasey & Daleiden, 1994).

Several recent studies have investigated the prevalence and phenomenology of panic attacks in children and adolescents (e.g., Hayward et al., 1992; King, Gullone, Tonge, & Ollendick, 1993). These studies suggest that, whereas only a small minority of young children experience isolated panic attacks, their incidence increases dramatically as children grow older. There is evidence to support the idea that children's interpretation of panic attacks changes as a function of cognitive development. While young children tend to relate panic symptoms to external causes, adolescents attribute panic symptoms to internal sources (Nelles & Barlow, 1988). It is only with such internal, catastrophic interpretations (e.g., "I am losing control", "I am going crazy") that a full-blown panic syndrome may develop. In contrast, external interpretations of panic symptoms may sustain a pathological radicalization of specific fears, a point to which we will return later.

Severity of Childhood Fears

It is far from clear how serious developmental fears are. In Ollendick and King's (1994) words: "Do children really worry about that many fears on a

frequent or regular basis? Do they engage in avoidance behaviors to prevent their occurrence? Do the reported fears interfere with their daily functioning?" (p. 635). Few studies have addressed these questions in a systematic fashion. In a survey by McCathie and Spence (1991), children were asked to complete the FSSC-R and the Fear Frequency and Avoidance Survey Schedule for Children (FFASSC). The FFASSC measures the frequency with which children respond with fearful thoughts and avoidance behavior to fear items listed by the FSSC-R. McCathie and Spence noted that there are robust connections between the most commonly reported fears on the FSSC-R and the frequency of fearful thoughts and avoidance behaviors. Thus, not only did children report having FSSC-R defined fears, they also said that these fears were accompanied by aversive thoughts and avoidance behavior. Likewise, Ollendick and King (1994) found that a large majority of children (more than 60%) reported that their fears interfered substantially with daily activities.

These findings emphasize the point that childhood fears are often seriously distressing. Yet, in order to get a better picture of the clinical significance of these fears, their connection to DSM-defined specific phobias and other anxiety disorders should be considered. A recent study by Muris, Merckelbach, Mayer, and Prins (2000) explored this issue. Fears of 290 children aged 8–13 years were assessed and then their severity was evaluated by means of a structured diagnostic interview measuring anxiety disorders in terms of DSM criteria. Results showed that in a sizeable minority of the children (22.8%), fears reflect significant anxiety disorders, notably specific phobias. There has been a strong tendency in the literature to portray childhood fears as mild and nonpathological phenomena (e.g., Rutter, Tizard, & Whitmore, 1968). While this may be true most of the time, the studies summarized in this section make clear that at least a subgroup of children evidence clinically significant and disabling phobias.

Childhood Specific Phobias

Epidemiological surveys have yielded prevalence rates of childhood specific phobias that vary between 2% and 9% (see, for reviews, Bernstein & Borchardt, 1991; Ollendick, Hagopian, & King, 1997). However, a recent study (Costello et al., 1996) that recruited nonclinical samples from the 9–13 years age range found markedly lower 3-months prevalence rates for specific phobias: 0.42% for girls and 0.13% for boys. Whatever the cause of these discrepancies in prevalence rates, there can be little doubt that specific phobias are one of the most commonly diagnosed anxiety disorders in children.

There is little consensus about the natural course of childhood specific phobias. Whereas there is evidence that a substantial proportion of these phobias remit without treatment (e.g., Agras, Chapin, & Oliveau, 1972), retrospective studies of adult phobic patients clearly demonstrate that specific phobias often have their onset in childhood (e.g., Öst, 1987). As a matter of fact, Thyer, Parrish, Curtis, Nesse, and Cameron (1985) found that

almost half of their large sample of patients with specific phobias reported an onset in the preteen years. In agreement with the latter position are the results of Strauss and Last (1993). These authors examined onset ages and other features of specific phobia in children referred for treatment services. The mean age of their sample was 11 years, while the mean onset age reported by this sample was 7.8 years, suggesting that specific childhood phobias have a relatively long history before they enter treatment programs.

Conflicting results about the developmental stability of childhood specific phobias may be more apparent than real. For example, Last, Perrin, Hersen, and Kazdin (1998) reported that 3 years after the initial diagnosis of an anxiety disorder, a large majority of clinically referred children and adolescents no longer met the criteria for their initial diagnosis. However, a substantial percentage of these "recovered children switched to a new anxiety disorder. This is an important finding because it suggests the presence of a general, higher order factor that predisposes to a broad spectrum of anxiety disorders. Last and associates also noted that separation anxiety disorder had the highest recovery rate (96%), whereas specific phobias were characterized by relatively low recovery rates (69%). A survey by Newman et al. (1996) demonstrates that, from childhood to early adulthood, anxiety disorder diagnoses become progressively more stable. Thus it is safe to assume that, while not all childhood specific phobias persist into adulthood, adult specific phobias often have their origins in childhood or early adolescence.

Overall, girls have higher scores on self-report measures of fear than do boys (e.g., Ollendick et al., 1989). Does this mean that they are at a higher risk for developing specific phobias? The answer seems to be affirmative. According to the DSM-IV, between 75% and 90% of the adults with animal, situational, or natural environment phobias are female. For blood-injection-injury phobia, the male–female distribution is somewhat more balanced: between 55% and 70% of the individuals with this type of phobia are female (Frederikson, Annas, Fischer, & Wik, 1996). As demonstrated by Muris, Schmidt, & Merckelbach (1999), the skewed sex distribution of specific phobia symptoms is also evident for children and adolescents.

In conclusion, then, specific fears are common in childhood and there are good reasons to believe that cognitive development is linked to the content of these fears. Furthermore, in most children, these fears represent transitory phenomena. Yet, in a subgroup, specific fears become chronic and develop into a phobia. The factors that are involved in this radicalization of childhood fears can be roughly grouped into genetically linked factors and environmental influences.

Genetically Linked Factors

Genetics

Specific fears and phobias run in families. This point is illustrated by the work of Fyer and colleagues (1990) who found that first-degree relatives (i.e.,

brothers and sisters) of probands with specific phobias more frequently suffered from specific phobias (31%) than relatives of control probands (11%). In addition, 15% of the children of the specific phobia probands were diagnosed with specific phobia, compared with 8% of the children of the control probands. Results also indicated that first-degree relatives of specific phobia probands did not have an increased risk for any psychiatric disorder other than specific phobia. On the basis of these findings, Fyer et al. (1990, p. 255) conclude that "specific phobia is a highly familial disorder that breeds true." This conclusion is further substantiated by recent work of Frederikson, Annas, and Wik (1997) who documented that spider and snake phobic women often report a positive familial history of such phobias.

In general, behavioral–genetic studies have shown clearly that genetic transmission contributes to the familial aggregation of specific fears and phobias. Rose and Ditto (1983) compared the frequency of self-reported fears in monozygotic and dizygotic twin pairs with ages ranging between 10 and 34 years. These authors found that a twin's level of fearfulness could be predicted from the co-twin's score. Also, the frequency of fears was more similar in monozygotic than in dizygotic twin pairs. By and large, these findings were replicated in a twin study of Stevenson, Batten, and Cherner (1992). These authors conducted analyses on FSSC-R scores of monozygotic and dizygotic twins in the 8–16 years age range and this led to two major conclusions. To begin with, whereas heritability (h^2) was significant for the summed FSSC-R scores, the specific FSSC-R components revealed a diverse picture, with some components having a nonsignificant heritability (e.g., medical fears) and others having a highly significant heritability (e.g., fear of injury and small animals: $h^2 = 0.46$). Second, no evidence was found to suggest that heritabilities increase at the extreme end of the fear continuum.

Taylor (1998) reviewed a number of large-scale behavioral–genetic studies concerned with phobias. Although this review was not particularly focused on childhood phobias, his differentiation between general and specific genetic factors is relevant to the present discussion. The general genetic factor acts as a vulnerability factor to a wide range of phobic fears, while specific factors predispose to certain types of fears. Taylor (1998, p. 211) summarized the role of both factors in phobic etiology as follows:

> The general factor tended to make a modest contribution to agoraphobia, situational phobia, and social phobia (7–10% of variance; mean = 9%) and a greater contribution to animal phobia (35%). Conversely, specific genetic factors were more important for agoraphobia, situational phobia, and social phobia (20–29%; mean = 23%) compared to animal phobia (0%).

Taylor points out that the general genetic component may constitute the biological substrate of what is typically referred to as "negative affectivity" or "neuroticism." Evidence for the idea that a general genetic factor contributes to anxiety via temperamental traits comes from a study by Zinbarg

and Barlow (1996). That study relied on a large sample of anxiety-disordered adults, including patients with specific phobias. Patients completed a battery of self-report scales selected to provide a broad coverage of the key symptoms of various DSM-defined anxiety disorders. Scores on these scales were subjected to factor analyses and this revealed a general higher order trait common to all anxiety disorder diagnoses. Further evidence for a general higher order trait is provided by Hofmann, Lehman, and Barlow (1997), who noted that having one specific phobia also increases the likelihood of experiencing subclinical fears from other phobia subtypes. An obvious behavioral approximation of this higher order trait is behavioral inhibition.

Behavioral Inhibition

Behavioral inhibition is the tendency of some children to interrupt ongoing behavior and react with vocal restraint and withdrawal when confronted with unfamiliar people or settings. Behavioral inhibition is thought to be a stable and inherited response disposition that characterizes approximately 10–15% of these children (e.g., Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). Cross-sectional and longitudinal data collected by Biederman and colleagues (1990, 1993) strongly suggest that this response disposition is a risk factor for anxiety disorders. These studies showed that preschool children identified as behaviorally inhibited are more likely to have anxiety disorders (including phobias) than control children. This became even more prominent at a 3-year follow-up: in the cohort of children who were initially identified as behaviorally inhibited, the rates of specific phobias and other anxiety disorders had increased markedly (see, for a review, Biederman, Rosenbaum, Chaloff, & Kagan, 1995). Thus, behavioral inhibition seems to be a vulnerability factor for a broad range of anxiety disorders, among which are specific phobias. It is not surprising, therefore, that it has been linked to higher order constructs such as neuroticism, trait anxiety (Craske, 1997), and negative affectivity (Clark, Watson, & Mineka, 1994).

In line with the results from behavioral inhibition studies, Stevenson-Hinde and Glover (1996) found that extreme shyness in preschool children is accompanied by relatively high levels of negative mood, worry, and fears. Behavioural inhibition, and in its wake shyness, are considered to be enduring temperamental traits that are relatively stable from early to middle and late childhood (Kagan, Reznick, Snidman, 1987, 1988; Scarpa, Raine, Venables, & Mednick, 1995; Gest, 1997). However, few studies have looked directly at the connection between behavioral inhibition/shyness and anxiety symptoms in older children. One exception is a recent study by Muris, Merckelbach, Wessel, and Van de Ven (1999). Children aged 12–14 years were provided with a definition of behavioral inhibition and then asked to classify themselves as low, middle, or high on behavioral inhibition. In addition, children completed questionnaires of worry, depression, and anxiety symptoms. Results showed that children in the high behavioral inhibition category had elevated levels of anxiety, worry, and depression compared with

children who endorsed the low or middle behavioral inhibition categories. Moreover, children who rated themselves high on behavioral inhibition more frequently exhibited anxiety disorder symptoms in the subclinical range than did children scoring low or intermediate on behavioral inhibition.

As to the biological underpinnings of behavioral inhibition, relevant parameters have been identified by Schmidt and coworkers (1997). These researchers noted that behaviorally inhibited children exhibit relatively high morning levels of the stress hormone cortisol. They speculated that high levels of cortisol may sensitize subcortical arousal circuits (e.g., amygdala, hypothalamus) and this would make children more prone to develop serious anxiety symptoms. Interestingly, work on psychophysiological parameters that tap subcortical fear responsivity (e.g., the eye blink startle reflex; Vrana, Spence, & Lang, 1988) support such an interpretation. For example, Grillon, Dierker, and Merikangas (1997) measured startle reflexes in children with a parental history of an anxiety disorder (who often meet the criteria for behavioral inhibition; see Rosenbaum et al., 1988) and control children. It was found that startle magnitude was relatively elevated in children of parents with an anxiety disorder. This is in agreement with the notion that anxiety-prone (i.e., behaviorally inhibited) children have hyperexcitable subcortical circuits that may promote fear behavior (see, for an extensive review, Rosen & Schulkin, 1998).

Another promising research area is concerned with the connection between frontal brain asymmetries and behavioral inhibition. A variety of evidence (see, for a review, Tomarken & Keener, 1998) indicates that the left frontal areas sustain approach behavior, while the right frontal areas are involved in avoidance behavior. This raises the possibility that a stable right frontal hyperactivation is a biological substrate of the tendency to react with withdrawal to potentially threatening stimuli. Germane to this issue is a study by Davidson and Fox (1989) who demonstrated that young infants with a strong activated right frontal hemisphere tend to react with crying to subsequent maternal separation. More recent studies suggest that relative right frontal hyperactivation in children is linked to a much broader response style that comes close to behavioral inhibition (e.g., Calkins, Fox, & Marshall, 1996). Perhaps, then, relative right frontal hyperactivation might be a good marker of behavioral inhibition and, possibly, severity of childhood specific phobias. While there have been some studies of EEG asymmetries in adult phobics (e.g., Davidson, 1998; Merckelbach, Muris, Pool, & De Jong, 1998), more research is required to delineate the precise connections between EEG frontal asymmetries, behavioral inhibition, and childhood specific phobias (see, for a critical review, Turner, Beidel, & Wolff, 1996).

Disgust Sensitivity

The studies reviewed above converge on the notion that behavioral inhibition affects a broad spectrum of phobic disorders. Thus, it qualifies as the behavioral expression of what Taylor (1997) termed a higher order genetic factor.

According to this author, lower order (i.e., more specific) genetic factors and their behavioral manifestations seem to play a less prominent role in specific phobias, but one possible exception in this context is disgust. Disgust has been identified as a food-related basic emotion, in part because it has clear biologically prewired antecedents. For example, young babies react with a spitting reflex when they are exposed to bitter substances (Ekman, 1992). Disgust sensitivity can be measured reliably with self-report scales that contain items like "It bothers me to see someone in a restaurant eating messy food with his fingers" (Haidt, McCauley, & Rozin, 1994). Psychometric studies have revealed that scores on such disgust scales are correlated positively with neuroticism and negatively with sensation seeking, although the correlations with these fundamental traits are modest (Haidt et al., 1994; Hennig, Pössel, & Netter, 1996).

Haidt and associates (1994, p. 711) concluded that "Disgust appears to make people cautious not only about what they put into their mouths, but about what they do with their bodies." Yet, recent findings suggest that the contribution of disgust sensitivity to anxiety is much more specific. There is now good evidence that disgust sensitivity is involved in the genesis of certain types of specific phobias, in particular animal phobias and blood-injection-injury phobias. For example, Matchett and Davey (1991; see also Merckelbach, De Jong, Arntz, & Schouten, 1993) found a positive association between measures of disgust sensitivity and scores on the animal phobia scale of the Fear Survey Schedule. A subsequent study (Davey, Forster, & Mayhew, 1993) not only noted a significant correlation between parents' and children's disgust scores, but also indicated that parental disgust sensitivity was the main predictor of offspring animal fear.

Only one study has investigated directly the role of disgust sensitivity in childhood animal phobia. De Jong, Andrea, and Muris (1997) assessed fear of spiders, disgust sensitivity, and spiders' disgust-evoking status in spider phobic girls who applied for treatment, in nonphobic girls, and in the parents of both groups of children. Phobic girls were tested twice, before and after behavioral treatment. The idea that disgust is an important aspect of spider phobia was supported by the following findings. To begin with, compared with control girls, spider phobic girls exhibited higher levels of disgust sensitivity and considered spiders per se as more disgusting. Second, after treatment, the reduction in spider fear was paralleled by a decline in spiders' disgust-evoking status. Third, mothers of spider phobic girls attributed a high disgust-evoking status to spiders. De Jong et al. (1997, p. 559) interpret the latter finding in terms of modeling experiences: "The acquisition of spider fear is facilitated by specific parental disgust reactions when confronted with spiders." Alternatively, it may well be the case that a genetic factor is involved in the familial transmission of disgust sensitivity and, in its wake, animal phobia.

As to the connection between disgust and blood-injection-injury (BII) phobia, Page (1994) has argued that elevated disgust sensitivity is responsible for the fainting component of BII phobia. Consistent with a genetic

interpretation are studies that report strong parent–child correspondence for fainting reactions to BII stimuli (e.g., Kleinknecht & Lenz, 1989). Unfortunately, no study has explored the links between disgust sensitivity and BII fear in children. Meanwhile, adult studies suggest that this may be a promising line of research. For example, Tolin, Lohr, Sawchuk, and Lee (1997) demonstrated that both spider phobic and BII phobic undergraduates exhibited elevated disgust sensitivity levels (see, for a similar finding, De Jong & Merckelbach, 1998). Interestingly, Tolin and colleagues found suggestive evidence that spider phobics react with a combination of fear and disgust to spiders, whereas BII phobics react with disgust rather than fear to medical stimuli.

Some authors (e.g., Phillips, Senior, Fahy, & David, 1998) have speculated that the basic emotion of disgust underlies a wide range of psychiatric symptoms. According to these authors, not only certain types of phobias, but also depression, eating disorders, and obsessive–compulsive disorder, would be related to disgust sensitivity. The evidence for such a broad relevance of disgust to psychopathology is, however, meager. In the final analysis, disgust can best be conceptualized as a rather specific factor.

Fear of Suffocation

Panic attacks are discrete episodes of intense fear accompanied by strong bodily sensations (e.g., shortness of breath, dizziness, sweating). Epidemiological surveys indicate that specific phobias are strongly comorbid with panic attacks (e.g., Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996). While DSM-IV descriptions of specific phobias portray panic attacks as secondary consequences of these conditions, evidence from various sources suggests that the role of panic attacks in the etiology of certain specific phobias may be more prominent than previously thought. There is, of course, older work demonstrating that panic attacks may constitute conditioning trials that produce persistent agoraphobic avoidance (e.g., Gorman, Liebowitz, Fyer, & Stein, 1989). Likewise, studies examining key cognitions of claustrophobia show that typical panic thoughts such as fear of suffocation, loosing control, and being trapped dominate this type of phobia and that successful treatment of claustrophobia depends critically on the extent to which these cognitions can be corrected (e.g., Shafran, Booth, & Rachman, 1993).

More recent work indicates an even closer connection between certain specific phobia types and panic. For example, Verburg, Griez, and Meijer (1994; see also Antony, Brown, & Barlow, 1997) exposed patients with animal phobias, patients with situational or natural environment phobias, and control subjects to a 35% carbon dioxide panic provocation challenge. These authors noted that patients with situational or natural environment phobias, but not animal phobics or controls, reacted with an increase in subjective anxiety that was similar to that found in earlier studies with panic patients. According to Verburg et al., situational/natural environment phobias are

characterized by excessive fear of suffocation, and panic-like experiences may be responsible for the onset of this category of specific phobias. By this view, isolated panic attacks are attributed by the patient to the context in which it occurred and this could result in a specific phobia. Consistent with such an interpretation are the findings of Himle, Crystal, Curtis, and Fluent (1991), who found that almost half of their sample of situational phobics ascribed the onset of their complaints to a panic-like experience in specific situations that subsequently became phobogenic (see also Merckelbach, De Ruiter, Van den Hout, & Hoekstra, 1989). Further support for the link between certain specific phobia types and panic is provided by the high incidence of natural environment and situational fears among panic disorder patients (De Ruiter, Rijken, Garssen, Van Schaik, & Kraaimaat, 1989; Starcevic & Bogojevic, 1997). In most cases, these specific fears and phobias precede the onset of panic disorder.

Exaggerated fear of suffocation is thought to reflect a biological and possibly heritable trait linked to what Klein (1993) has termed "hypersensitive suffocation detectors." According to Klein, these hypothetical brain detectors trigger panic attacks in response to a broad range of stimuli that may signal a lack of useful air (i.e., suffocation). Support for this conceptualization comes from the finding that respiratory abnormalities in response to challenges occur more often in anxiety disordered children than in controls (Pine et al., 1998), from studies showing that the incidence of anxiety disorders (among which severe specific phobias) is considerably heightened in children with asthma (Bussing, Burket, & Kelleher, 1996), and from a medical condition known as "congenital hypoventilation syndrome" (Ondine's curse) in which children have a deficient suffocation detector and evidence fewer anxiety symptoms (Pine et al., 1994). To the extent that the role of hypersensitive suffocation detectors in situational/environmental phobias is genetically determined, it can be considered as a specific genetic contribution. On the other hand, hypersensitive suffocation detectors trigger isolated panic attacks and it is these panic attacks that are believed to set up conditioning trials leading to phobic avoidance (Forsyth & Eifert, 1996). Thus, panic attacks may also be conceived as learning experiences, an issue to which we turn now.

Environmental Influences

By and large, twin studies have shown that genetic factors play a significant but modest role in the etiology of specific phobias. Most of these studies have come up with heritability estimates for specific phobias that are considerably lower than those for, say, bipolar disorders or schizophrenia. For example, Kendler et al. (1992, p. 279) conclude from their analysis of twin data that "the estimated heritability of liability for phobias (...) indicates that genetic factors play a significant but by no means overwhelming role in the etiology of phobias." A similar conclusion can be drawn from studies concerned with

behavioral inhibition in children. About 70% of the children classified as behaviorally inhibited remain free of any anxiety disorder (Biederman et al., 1990). Apparently, then, environmental factors determine whether genetically transmitted vulnerabilities culminate in specific phobias.

Following Taylor (1998), one may differentiate between general environmental factors contributing to all phobias and specific environmental factors unique to each type of phobia. Negative life events and dysfunctional parental rearing behavior constitute such a general factor, while discrete learning experiences correspond to specific environmental factors.

Negative Life Events

Several studies have found an increased incidence of negative life events such as parental divorce or death of significant family members in clinically anxious children (Benjamin, Costello, & Warren, 1990; Goodyer, Wright, & Altham, 1990; Kashani et al. 1990). However, it is unlikely that these life events per se are responsible for the emergence of phobias or other anxiety disorders. Spence and Dadds (1996) argue that the negative impact of aversive life events critically depends on factors that either exacerbate (e.g., behavioral inhibition; cf. *supra*) or buffer (e.g., social support and effective coping strategies) their effects. Interestingly, Hekmat (1987) found some evidence to suggest that environmental stressors produce a larger fear increase in individuals with traits that are related to neuroticism.

A more articulated theory about the contribution of negative life experiences to the etiology of specific phobias was proposed by Jacobs and Nadel (1985). These authors noted that developmental fears may reappear after exposure to stressful events. According to the Jacobs and Nadel model, the slowly maturing hippocampus is responsible for contextual control and inhibition of developmental fears. However, stressful conditions would disrupt hippocampal functioning and this would result in the re-emergence of context-free and stereotyped fear responses that are typical for early maturational stages. While the Jacobs and Nadel theory offers an interesting perspective on the role of negative life events in the radicalization of developmental fears, direct empirical evidence supporting this model is lacking (see, for a critical review, McNally, 1989).

Parental Rearing Behaviors

A number of studies have examined the role of parental rearing behaviors in the development of anxiety disorders (e.g., Rapee, 1997). Basically, these studies have followed one of two research lines. The first line is inspired by attachment theory and focuses on early parent–child interactions. The second line is based on a broader perspective and examines the connection between ongoing parental rearing behaviors (i.e., parental rearing styles) and anxiety.

Attachment theory proposes that children's level of anxiety is affected by the way in which they are attached to their caregivers (Bowlby, 1973). There is, indeed, evidence to suggest that early attachment relationships are connected to fear and anxiety in later childhood. For example, in their prospective study, Warren and colleagues (1997) classified infants as either securely or insecurely attached. When children reached 17.5 years of age, current and past anxiety disorders were assessed by means of an interview schedule. Insecurely attached children more frequently suffered from anxiety disorders than control children. Unfortunately, Warren and associates did not include specific phobias in their analyses. A recent study by Muris, Mayer, and Meesters (2000) further examined the connection between attachment styles and anxiety disorder symptomatology. Children were asked to complete Hazan and Shaver's (1987) single-item measure of attachment style and the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997; Muris, Merckelbach, Schmidt, & Mayer, 1999), an index of DSM-defined anxiety disorder symptoms. In accordance with Bowlby's position, insecurely attached children exhibited elevated levels of anxiety disorder symptomatology compared with securely attached children. However, securely and insecurely attached children did not differ with regard to specific phobia symptoms. Thus, while insecure attachment seems to promote the development of the more complex anxiety disorders (e.g., generalized anxiety disorder), there is no direct evidence that this factor also plays a major role in the etiology of specific phobias.

While attachment researchers emphasize that disturbances in early parent–child interactions promote the development of high anxiety levels in children, others have stressed the importance of particular parental rearing styles. Evidence for this position comes from two classes of studies. The first relies on direct observation of current parent–child interactions. Following such an approach, Dadds and colleagues (Dadds, Barrett, & Rapee, 1996; see, for a review, Dadds & Barrett, 1996) noted that parents of anxious children often encourage their children to rely on avoidant coping strategies. Similarly, a study by Muris, Steerneman, Merckelbach, and Meesters (1996) demonstrated that specific fears reported by the children are a function of the extent to which mothers express their own fears in the presence of their children. A second class of studies makes use of questionnaires that intend to measure children's perceptions of parental rearing behaviors. For example, in a study by Muris and Merckelbach (1998a), normal schoolchildren completed the child version of the EMBU (Egna Minnen Betraffende Uppfostran [My memories of upbringing]; Castro et al. 1993), a questionnaire that taps children's perceptions of four main dimensions of parental rearing behaviors: emotional warmth, rejection, control, and anxious rearing. Additionally, children completed the SCARED (cf. *supra*). Results revealed significant and positive relationships between, on the one hand, anxious rearing behaviors and parental control, and, on the other hand, anxiety disorder symptomatology—in particular, symptoms of

generalized anxiety disorder, separation anxiety disorder, and to a lesser degree situational/natural environment phobia.

Specific Learning Experiences

According to Rachman's (1977, 1991) influential three-pathways theory, there are three types of discrete learning experiences that may play a role in the acquisition of phobias: (1) aversive classical conditioning; (2) modeling (vicarious learning); and (3) negative information transmission (exposure to negative information about the phobic object). Rachman (1977) further speculated that severity of fears is a function of acquisition mode. More specifically, fears acquired through vicarious learning or exposure to negative information would be less intense, whereas clinical phobias would be more likely to have a direct conditioning etiology.

Several studies have sought to test Rachman's three-pathways theory (see, for reviews, Menzies & Clarke, 1994; Merckelbach, De Jong, Muris, & Van den Hout, 1996; King et al., 1998; Muris & Merckelbach, 1998b). In most of these studies, adult phobics were asked to what extent the three pathways contributed to the onset of their complaints. Overall, these studies found some (but by no means unequivocal) support for Rachman's hypothesis that direct conditioning is the predominant pathway in clinical phobias, while indirect pathways (modeling and exposure to negative information) more frequently figure in the etiology of mild fears (Öst, 1991). One problematic feature of these findings is that they were based on patients who were asked to assign their pertinent learning experiences to the three pathways 10–20 years after the onset of their specific phobias (e.g., Menzies, Kirkby, & Harris, 1998). While it is true that the unreliability of retrospective reports should not be exaggerated (Brewin, Andrews, & Gotlib, 1993), support for the three-pathway theory would be stronger if it came from children and adolescents who were closer to the onset of their specific fears or phobias.

A number of recent studies have interviewed parents about the etiological pathways of their children's phobias or fears, whereas others have explored etiological routes to childhood specific fears and phobias by asking children directly about their learning experiences. The main findings of both categories of studies are listed in table 16.1.

Graham and Gaffan (1997) compared water-fearful children with nonfearful controls and had mothers of both groups complete a child fear origins questionnaire. Most mothers (78%) of fearful children said that the child's fear had always been present, but they also reported exposure to negative information and, to a lesser extent, aversive modeling and conditioning experiences. Remarkably, such learning experiences were also described by mothers of nonfearful children. Thus, these findings suggest that learning experiences of the sort implicated in Rachman's three-pathway theory are not so much responsible for the origins of fears, but rather play a role in their persistence and radicalization.

Table 16.1. Origins of Childhood Specific Fears and Phobias: Type of Fear, Sample Sizes, and Main Results

Study	Fear/phobia	Sample	Phobic origins	
Parent-based attributions				
Graham & Gaffan (1997)	Fear of water	9	Conditioning	22%
			Modeling	11%
			Negative information	78%
			Don't know	22%
Milgrom et al. (1995)	Dental fear	174	No percentages specified	
Menzies & Clarke (1993)	Water phobia	50	Conditioning	2%
			Modeling	26%
			Negative information	14%
			Always been afraid	56%
King et al. (1997)	Dog phobia	30	Conditioning	27%
			Modeling	53%
			Negative information	7%
			Don't know	13%
Child based attributions				
Ollendick & King (1991)	10 common FSSC-R fears	1092	Conditioning	37%
			Modeling	56%
			Negative information	89%
Muris, Merckelbach, & Collaris (1997)	Top intense fears	192	Conditioning	61%
			Modeling	50%
			Negative information	88%
Doogan & Thomas (1992)	Fear of dogs	11	Conditioning	91%
			Modeling	73%
			Negative information	82%
Merckelbach, Muris, & Schouten (1996)	Spider phobia	22	Conditioning	41%
			Modeling	14%
			Negative information	5%
			Always been afraid	46%
Merckelbach & Muris (1997)	Spider phobia	26	Conditioning	23%
			Modeling	8%
			Negative information	4%
			Always been afraid	62%

Note. In most studies, percentages do not add up to 100% since youths and/or parents could endorse more than one pathway.

In their large-scale study on the origins of childhood dental fear, Milgrom, Mancl, King, and Weinstein (1995) did not specify exact frequencies of conditioning, modeling, and negative information experiences in the subsample of fearful children. However, information about potential learning experiences provided by the mothers was subjected to a regression analysis and this led the authors to conclude that "both direct conditioning and parental modeling factors were significant independent predictors of fear level even when controlling for gender, age, and other sociodemographic

and attitudinal factors" (p. 318). At the very least, this suggests that an analysis of childhood fears in terms of Rachman's three-pathways is informative.

Menzies and Clarke (1993) as well as King, Clowes-Hollins, and Ollendick (1997) focused on clinically significant fears. Like Graham and Gaffan (1997), Menzies and Clarke found that the majority of parents (56%) believed that their child's water phobia had always been present. Even so, modeling episodes, exposure to negative information, and to a lesser extent conditioning experiences were reported as either "most influential" (modeling) or "somewhat influential" (negative information) factors in the etiology of their child's phobia. Remarkably similar findings were reported by King et al. In their study, parents endorsed modeling as the most important factor contributing to the development of their child's dog phobia, while very few parents endorsed exposure to negative information as the major pathway.

The studies reviewed so far were based on parental attributions, but as King et al. (1997, p. 77) rightly point out, reliance on parental perceptions of fear acquisition "may be quite invalid in terms of what actually occurred." In what seems to be the largest study on origins of childhood fears, Ollendick and King (1991) evaluated to what extent Rachman's theory of fear acquisition can be applied to the top 10 intensive FSSC-R fears. Children who reported "a lot" of fear to FSSC-R items such as "not being able to breathe", "being hit by a car or truck", and so forth, were given a short questionnaire that asked them whether they had experienced conditioning, modeling, and/or informational events related to these stimuli or situations. The authors found that a majority of the children (88.8%) attributed their fear to negative information. Modeling and conditioning events were less often mentioned by the children (56.2% and 35.7%, respectively).

In an attempt to replicate these findings, Muris, Merckelbach, and Collaris (1997) had children specify their top intense fear and then asked them whether conditioning, modeling, and negative information played a role in that fear. Like Ollendick and King (1991), these authors found that exposure to negative information was the most prominent pathway to fear mentioned by the children, followed by conditioning and modeling. In the Muris et al. study, children were also explicitly asked to what extent these learning experiences intensified their fears. Thus, whereas Ollendick and King (1991) employed broad definitions of the three types of learning experiences ("Did it play a role?), children in the Muris et al. study answered the additional question about whether these experiences served as antecedents of a radicalization of their fear. With this more strict definition, 46% of the children endorsed a conditioning pathway, while modeling and negative information were less often mentioned (4% and 35%, respectively).

Consistent with this are the findings of Doogan and Thomas (1992) who found that a large majority of dog-fearful children reported aversive conditioning encounters with dogs. However, the frequencies with which dog-fearful children reported such experiences did not differ from those found

in a group of nonfearful children, a finding that parallels the results of Graham and Gaffan (1997). Apparently, then, these learning experiences are not the primary cause of fear.

In a study by Merckelbach, Muris, and Schouten (1996), children with a severe spider phobia were interviewed about conditioning events, modeling experiences, and negative information transmission. To evaluate the reliability of the information provided by the children, parents were interviewed independently about the origins of their child's phobia. Conditioning events were reported by a substantial proportion of spider phobic children and a majority of these events were confirmed by their parents. Interestingly, nearly half of the children (46%) said that they had always been afraid of spiders, but even in this subsample, reports of conditioning and modeling experiences were found. These results were largely replicated by Merckelbach and Muris (1997) who also found a relatively high frequency of conditioning reports that were substantiated by parents.

Three important conclusions can be drawn from the studies reviewed in this section. To begin with, Rachman's three-pathway model is a valuable framework for conceptualizing the role of learning experiences in the development of childhood fears and phobias. Second, the results of clinical studies (e.g., Menzies & Clarke, 1993; King et al., 1997; Merckelbach, Muris et al., 1996) as well as analogous studies (e.g., Ollendick & King, 1991; Doogan & Thomas, 1992; Graham & Gaffan, 1997; Muris, Merckelbach, & Collaris 1997) suggest that conditioning and modeling are more important factors in the etiology of severe fears than negative information transmission. Third, given the high percentage of fearful children who claim to have always been afraid, and the high percentage of nonfearful children who claim to have experienced conditioning and/or modeling events, it is highly unlikely that the learning experiences implicated in Rachman's three-pathways model represent simple etiological antecedents of specific childhood fears and phobias. Rather, research findings are consistent with the idea that in some children such experiences have a stronger impact and thus contribute to a transition of normal, developmental fears into persistent phobias. Note, in passing, that there is a huge literature on personality traits and conditionability, showing that individuals with traits related to neuroticism or behavioral inhibition acquire fear responses with relative ease compared with individuals without such traits (e.g., Eysenck, 1987; see also Gershuny & Sher, 1998).

Maintenance of Fears and Phobias

How are specific fears and phobias maintained, once they have been acquired? The influential two-stage model of Mowrer (1960) suggests that avoidance behavior is responsible for the conservation of phobic fear. More specifically, avoidance would minimize direct and prolonged contact with the fear-provoking stimulus, and, hence, the phobic person would not have the opportunity to learn that it is in fact harmless.

While the role of avoidance behavior in the maintenance of fears and phobias seems self-evident, there are other mechanisms that are highly relevant in this context. Inspired by cognitive psychology, recent studies have sought to elucidate information-processing abnormalities that may play a critical role in the maintenance of anxiety symptoms (see, for reviews, Eysenck, 1992; McNally, 1998). These abnormalities have been termed "cognitive biases" and two of them may be especially relevant for understanding the persistence of specific phobias in children: attentional bias and interpretation bias.

Attentional Bias

A large number of studies have documented that adult phobics display hyperattention toward potentially threatening material. A frequently employed technique for demonstrating this attentional bias is the emotional Stroop task. In that task, subjects are required to name the color in which words are printed while ignoring the meaning of these words. A consistent finding in Stroop studies with, for example, spider phobics is that their color naming of threatening words (e.g., "creepy", "hairy") is slower than that of neutral words (e.g., "shady", "cars"). This is due to the fact that phobics automatically direct their attention to the content of the threatening words and this interferes with their main task of color-naming (e.g., Watts, McKenna, Sharrock, & Trezise, 1986). There are reasons to believe that learning experiences like, for example, aversive conditioning events promote an attentional bias (Merckelbach, Van Hout, De Jong, & Van den Hout, 1990). Also, attentional bias for threat cues disappears in phobics who have been successfully treated (e.g., Watts et al., 1986; Lavy, Van den Hout, & Arntz, 1993). Taken together, these findings indicate that the attentional bias phenomenon is a consequence rather than a cause of anxiety (McNally, 1998). However, this is not to say that attentional bias is an epiphenomenon without clinical ramifications. It is highly plausible that the attentional bias phenomenon leads to an increased encoding of threatening material, thereby elevating fear levels, which in turn intensifies the attentional bias etc. In Mineka and Sutton's (1992), words: "One can easily see how the attentional bias toward threatening information associated with anxiety would tend to perpetuate or even enhance the emotion because of the increased focus on danger and threat cues" (p. 68).

So far, most studies on attentional bias were concerned with adult patients and their symptomatology. Yet, there is evidence that the attentional bias phenomenon also occurs in fearful children. For example, in their Stroop experiment, Martin, Horder, and Jones (1992) found that spider-fearful children exhibited retarded color-naming times when confronted with spider-related words (e.g., "web"), but not when confronted with neutral words (e.g., "fly"). Likewise, using the dot-probe paradigm, Vasey and colleagues (Vasey, Daleiden, Williams, & Brown, 1995; Vasey, El-Hag, & Daleiden, 1996) demonstrated that clinically anxious children and children

high in test anxiety were faster to react to a probe if it was preceded by a threatening rather than a neutral word. This differential reaction reflects selective attention to threatening stimuli and was not evident for the control children in the Vasey et al. studies.

Thus, studies on attentional bias in fearful children have produced findings that closely parallel those reported in the adult literature. Yet, there is one exception that is worthy of note. In a series of studies, Kindt and colleagues (Kindt, Bierman, & Brosschot, 1997; Kindt, Van den Hout, De Jong, & Hoekzema, in press) examined response latencies of spider-fearful and control children aged 8–12 years in an emotional Stroop task. These researchers found consistently that attentional bias for spider-related material was not restricted to the highly fearful group, but also emerged in the control group. Furthermore, in the control group, age was found to be negatively correlated with attentional bias, suggesting a decline of attentional bias for spider-related words with age. In contrast, in the phobic group, a positive correlation was found between age and attentional bias, suggesting an increase of bias with age. On the basis of these data, Kindt et al. argue that at early developmental stages, attentional bias for threatening material may be a pervasive and normal phenomenon. As children grow older, they would learn to inhibit selective processing of threat cues. By this view, the development of childhood fears into phobias would be accompanied by a failed inhibition of selective attention. Such an interpretation fits nicely with the well-documented fact that, in most young children, normal developmental fears dissipate.

Interpretation Bias

Attentional bias for threat may be viewed as a fundamental characteristic that operates at the early stages of information processing. Yet, studies on anxious adults have revealed that there are other cognitive biases that operate at the more conceptual stages of information processing. A good example is provided by the interpretation bias. For example, Hadwin, Frost, French, and Richards (1997) confronted children low or high on trait anxiety with ambiguous homophones that had either a neutral or a threatening interpretation (e.g., dye vs. die). High levels of trait anxiety were found to be positively associated with threatening interpretations of homophones. In a study by Barrett, Rapee, Dadds, and Ryan (1996), anxious and nonanxious children received more complex stimulus material, namely ambiguous stories, and were then instructed to interpret them. Results indicated that anxious children were more likely to interpret ambiguous situations in a threatening way compared with nonanxious children. Similar findings were reported by Bell-Dolan (1995) and Chorpita, Albano, and Barlow (1996). The latter study also obtained some preliminary evidence that child–parent interactions might reduce a child's tendency to interpret ambiguous material as threatening.

All in all, it is fair to conclude that the interpretation bias of anxious children is a robust empirical phenomenon. In this respect, the findings are

highly similar to those found in the adult literature. Yet, no study has carried out a more thorough analysis of the specificity of this bias. Thus, it remains unclear whether this bias is linked to general characteristics such as trait anxiety or to specific anxiety states.

Conclusion

Our knowledge of factors that are involved in the etiology of childhood specific phobias has increased considerably. The present chapter has made an attempt to structure this knowledge using a tentative model that synthesizes concepts and evidence from various sources. Figure 16.1 presents an outline of the model. It emphasizes a multifaceted etiology of childhood specific phobias and is based on the assumption that there is a continuity between normal developmental fears and childhood specific phobias. However, whereas childhood fears are common, but transitory phenomena in most children, there is a small subgroup of children in whom these fears tend to radicalize owing to a genetic vulnerability. This genetic vulnerability

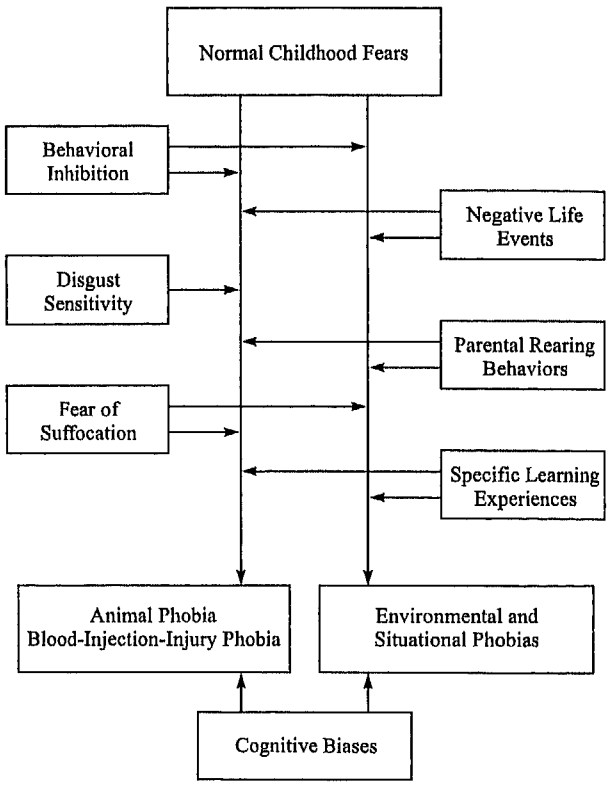

Figure 16.1. A multifactorial model of the etiology of childhood specific phobias.

may manifest itself in certain behavioral patterns (e.g., behavioral inhibition or disgust sensitivity). Stressful life events, parental rearing behaviors, and specific learning experiences (i.e., conditioning, modeling, and negative information) interact with normal developmental fears and genetically based behavioral patterns to produce extremely persistent fears that ultimately take the form of a specific phobia (or another anxiety disorder; see Craske, 1997). Once a specific phobia exists, it is maintained by cognitive mechanisms such as attentional bias.

We do not claim that the present model has the qualities of a scientific model in the strict sense of the word. Clearly, it lacks specificity. Most importantly, the precise dynamics between the various factors that figure in the model are far from clear. For example, do aversive learning experiences like conditioning or modeling contribute to a radicalization of developmental fears only during a critical period (e.g., when a certain developmental fear is at its maximum)? Or is it the case that learning experiences in combination with behavioral inhibition or disgust sensitivity may reinstate developmental fears that disappeared during a previous phase? These issues require longitudinal prospective studies that include various parameters. Of course, such complex prospective studies are not easy to conduct, precisely because they are time-consuming and difficult to get funded. But as Clark et al. (1994, p. 113) point out:

> Until prospective studies have followed individuals—whose premorbid personality and environmental characteristics are known—over sufficiently long periods of time to yield a sufficient base rate of disorder, we will not be able to distinguish causal from concomitant, confounding, or residual factors.

Not only prospective studies, but also comorbidity studies could make a major contribution to our understanding of the etiology of childhood specific phobias. For example, the connection between cognitive maturation and specific phobias can be worked out by examining fear symptoms in samples with clearly defined cognitive dysfunctions. Autistic disorder is a case of point (Muris, Steerneman, Merckelbach, Holdrinet, & Meesters, 1998): children with pervasive developmental disorder not otherwise specified display more frequently specific phobia symptoms than do children with autistic disorder, and this may well have something to do with higher IQ levels in the former group. A closer analysis of such associations would be informative. Much the same is true for the connection between isolated panic attacks and environmental/situational fear symptoms in children with medical conditions like asthma (Bussing et al., 1996).

Our multifaceted model is difficult to reconcile with the so-called nonassociative account of phobic etiology (Menzies & Clarke, 1993, 1995). The basic assumption of this account seems to be that discrete learning experiences play a marginal role in the etiology of specific phobias. According to the nonassociative account, common developmental fears reflect innate and spontaneous reactions to evolutionary prepotent cues. Specific phobias

would echo these spontaneous fear reactions. But, if this is the case, one might ask why not all children suffer from specific phobias. Menzies and Clarke (1993, 1995) propose that "poor habituators may remain fearful of innate fear cues. In these individuals, developmental" fears would become chronic and take the form of a specific phobia. Alternatively, nonspecific stressors (e.g., life events) could produce dishabituation and the reinstatement of developmental fears. In either case, specific phobias would originate directly from developmental fears which, in turn, would derive from innate responses to evolutionary dangers. This would also explain why adult patients with specific phobias often report that they have always been afraid of the phobic stimulus.

The nonassociative account is flawed on several counts. To begin with, it assumes that one can relate developmental fears to evolutionary challenges. Admittedly, it is relatively easy to construct an evolutionary scenario for, say, fear of heights or fear of strangers. But consider fear of spiders. In that case, an evolutionary interpretation is more problematic given the fact that only 0.1% of the 35,000 known spider varieties are dangerous. A second point is that the nonassociative account fails to explain *why* some individuals habituate poorly to prepotent fear stimuli. Furthermore, from a scientific point of view, it is not satisfactory to think of phenomena as "spontaneous" or "nonassociative." After all, science is about causal associations, so one would like to know where "spontaneous" phobias originate from (see, for critical reviews of the nonassociative account, Merckelbach & De Jong, 1997; Forsyth & Chorpita, 1997). In other words, etiological models of specific phobias cannot do without the explanatory power of concepts like disgust sensitivity, behavioral inhibition, and aversive conditioning.

The multifactorial model outlined in this chapter draws on extensive empirical work, and in many respects it approaches a consensus among researchers about the complexity of the etiological antecedents involved in phobias. Over the past years, the extensive research in this domain has, indeed, produced a high degree of theoretical sophistication. Paradoxically, theoretical refinements may create a certain risk that researchers or clinicians become attracted to more cavalier-like conceptualizations. This is nicely illustrated by a recent Dutch book on phobias that reintroduces the Freudian idea that specific phobias are symbolic references to internal but unconscious conflicts (Fuldauer, 1998). The author claims, for example, that phobias of spiders would reflect a problematic marriage while phobias of frogs derive from conflict with parents. As another example, recent years have seen the rapid proliferation of power therapies like eye-movement desensitization and reprocessing (EMDR; see, for a critical analysis, Rosen, Lohr, McNally, & Herbert, 1998). These therapies have been promoted as fast cures for all sorts of anxiety disorders, among which are specific phobias. Controlled outcome studies (e.g., Muris, Merckelbach, Sijsenaar, & Holdrinet, 1998) have shown that EMDR adds nothing to the well-established exposure-based therapies. Apparently, theoretical advances may be accompanied by professional ignorance, and this underlines that it is

important for professional organizations to encourage consensus development and research-based treatment protocols.

Note

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